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The role of catecholamines in aneurysmal subarachnoid hemorrhage: a narrative review

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The marked release of catecholamines during subarachnoid hemorrhage is an important aspect of the pathobiology following aneurysmal rupture. This narrative review aims to identify how catecholamines influence aneurysmal subarachnoid hemorrhage (aSAH) outcomes. aSAH is a critical neurological condition characterized by hemorrhage into the subarachnoid space, leading to severe neurological deficits and mortality. Catecholamines, including epinephrine, norepinephrine, and dopamine, are the body's stress responses, which can lead to secondary injuries following aSAH. This review was conducted through a targeted literature search of relevant studies examining the relationship between aSAH, catecholamines, and clinical outcomes. Searches were performed in PubMed, Scopus, The Cochrane Library, Medline (Ovid), Embase (Ovid), and CINAHL, including publications up to July 2024. Search terms combined keywords and subject headings related to "subarachnoid hemorrhage" or "aSAH," "catecholamines," "epinephrine," "norepinephrine," "dopamine," and outcome-related terms such as "prognosis," "mortality," and "neurological outcome." Articles were selected based on relevance, and key findings were synthesized descriptively to provide a comprehensive overview of current knowledge in this area. Elevated levels of catecholamines are observed following aSAH and are associated with increased sympathetic nervous system activity. This catecholamine surge contributes to pathological processes, including vasospasm, blood-brain barrier disruption, cerebral edema, and neuronal damage. The review highlights the implications of catecholamine levels; where higher concentrations correlate with poorer outcomes and higher mortality rates. Understanding the mechanisms responsible for secondary injury due to catecholamines surge following aSAH shall facilitate the development of therapeutic approaches to prevent secondary brain injury and improve outcomes.

Key Words: blood-brain barrier; catecholamines; cerebral edema; dopamine; epinephrine; norepinephrine; secondary injury; subarachnoid hemorrhage; vasospasm

Review Article

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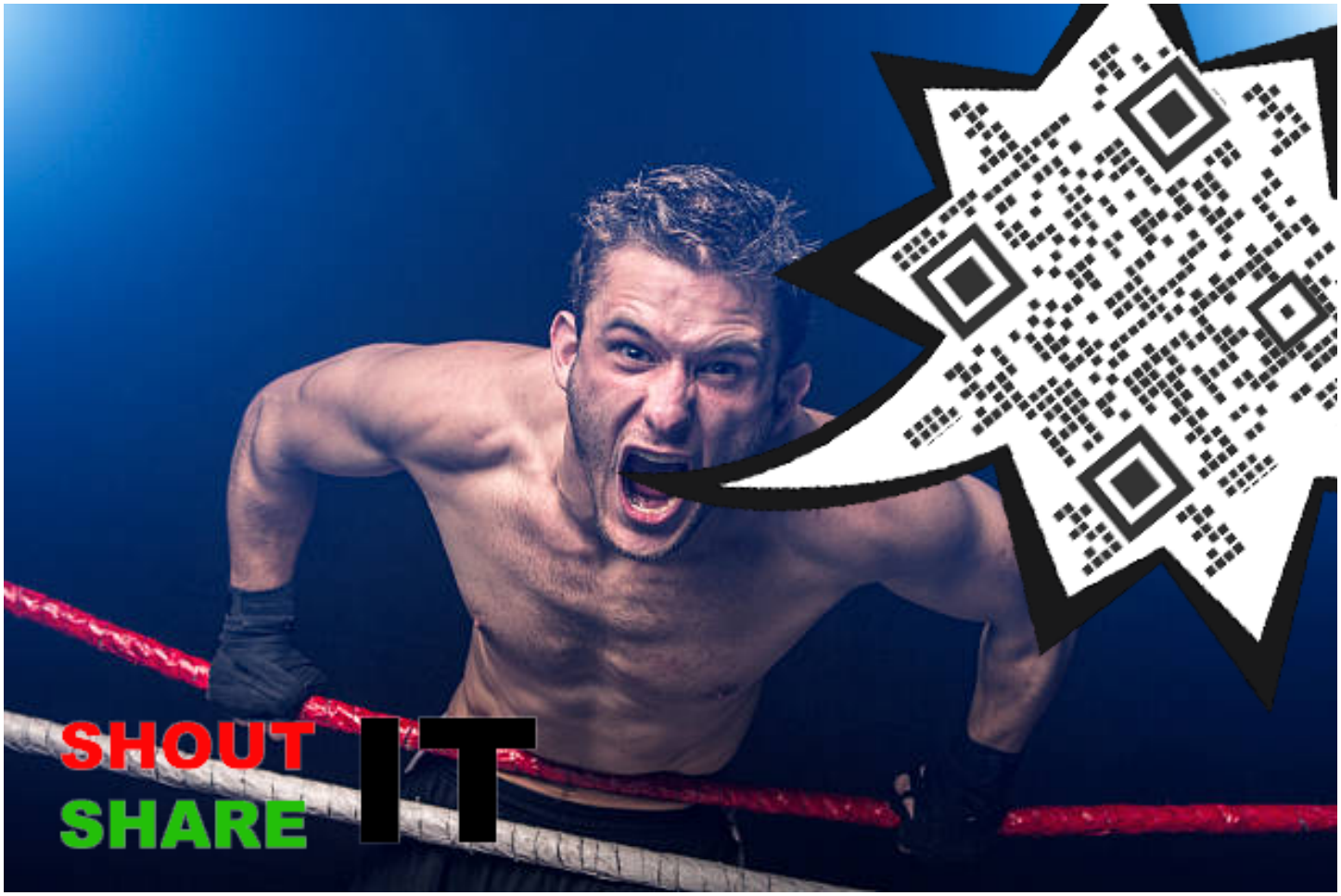
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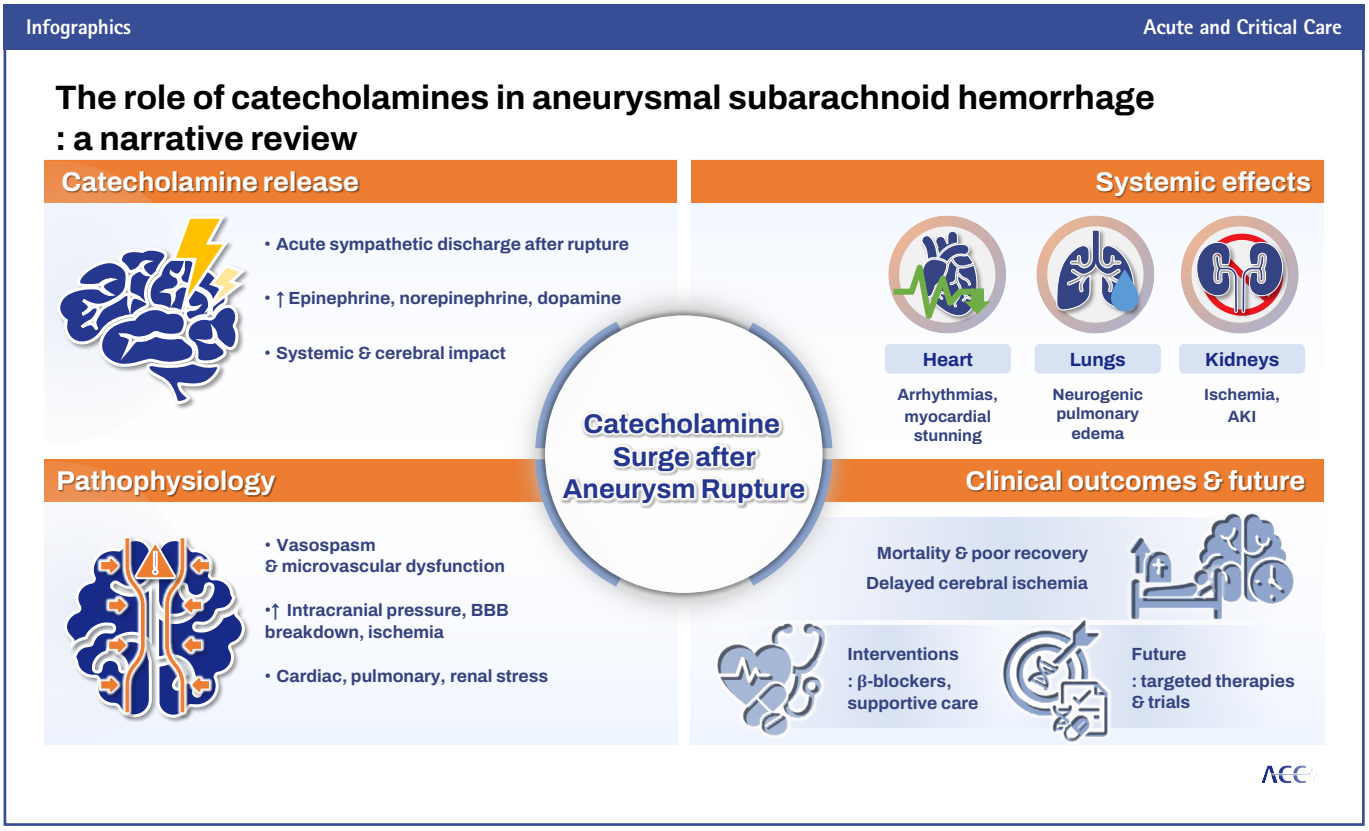

INTRODUCTION

Aneurysmal subarachnoid hemorrhage (aSAH) is characterized by hemorrhage into the subarachnoid space, often resulting from a ruptured cerebral aneurysm. aSAH not only causes neurological deficits, increased morbidity, and mortality rates but also results in the release of catecholamines (i.e., adrenaline, noradrenaline, and dopamine). Intracranial aneurysms represent common vascular lesions, and their risk of rupture depends on various morphological and anatomical factors [1,2]. Features such as larger size, a higher aspect ratio, and a location in the posterior circulation have been shown to significantly increase rupture probability [2]. When rupture occurs, subarachnoid hemorrhage ensues, leading to considerable morbidity and a reported case fatality rate greater than 30% [1]. Following aneurysmal rupture, a massive catecholamine surge occurs due to sympathetic nervous system activation and hypothalamic-pituitary-adrenal axis stimulation. This catecholamine excess contributes to secondary brain injuries, including early brain injury and delayed cerebral ischemia (DCI), through vasoconstriction, microthrombosis, and blood-brain barrier (BBB) disruption. Additionally, catecholamine surges can increase cerebral metabolic demand and contribute to oxidative stress, exacerbating neuronal injury [3]. Systemically, they can lead to complications such as neurogenic stunned myocardium and neurogenic pulmonary edema (NPE), which further complicate clinical management and can impact overall outcomes. Elevated catecholamine levels have been associated with poor clinical outcomes in aSAH, underscoring the importance of understanding their role in its pathophysiology and management [4]. This review explores the current understanding of how catecholamines influence the pathophysiology, secondary brain and systemic injuries, and clinical outcomes of aSAH.

METHODOLOGY

This narrative review was conducted through a targeted literature search of relevant studies addressing the relationship between subarachnoid hemorrhage (aSAH), catecholamines, and clinical outcomes. Key electronic databases searched included PubMed, Scopus, The Cochrane Library, Medline

(Ovid), Embase (Ovid), and CINAHL, with searches performed up to July 2024. Search terms consisted of combinations of keywords and subject headings related to "subarachnoid hemorrhage" or "aSAH," "catecholamines," "epinephrine," "norepinephrine," "dopamine," and outcome-related terms such as "prognosis," "mortality," and "neurological outcome." The search strategy was designed to capture a broad range of relevant literature, including clinical trials, observational studies, and review articles. Selected articles were chosen based on their relevance to the topic, and key findings were synthesized descriptively to provide a comprehensive overview of current knowledge in this area.

KEY MESSAGES

- Elevated levels of catecholamines are observed following aneurysmal subarachnoid hemorrhage and are associated with increased sympathetic nervous system activity.
- The catecholamine surge drives pathological processes, such as vasospasm, blood-brain barrier disruption, cerebral edema, and neuronal damage.
- Higher catecholamine concentrations are associated with poorer outcomes and increased mortality rates.

PATHOPHYSIOLOGY OF CATECHOLAMINES IN SAH

Initial Surge and Hemodynamic Instability

Following the rupture of an aneurysm, there is a rapid and substantial release of catecholamines. This surge is primarily due to the activation of the sympathetic nervous system in response to sudden stress and pain. Elevated catecholamine levels lead to increased blood pressure and heart rate, which can exacerbate the bleeding and worsen the initial brain injury. Cruickshank et al. [5] demonstrated that in SAH, elevated catecholamine levels can contribute to electrocardiographic changes, including ST-segment deviations and T-wave inversions, as well as myocardial damage characterized by myocardial necrosis and contraction band necrosis. These effects may be exacerbated by elevated corticosteroid activity, which increases cardiovascular sensitivity to catecholamines and can intensify cardiac dysfunction during the acute phase of SAH [5].

Vasospasm and DCI

Catecholamines surge leads to cerebral vasospasm and thus reducing blood flow to the brain. Vasospasm typically occurs between 4 to 14 days post-SAH. It is a significant cause of poor neurological outcomes and DCI, which is seen in up to 66% of individuals diagnosed with aSAH [6]. DCI includes emergence of focal motor deficits, changes in cognitive function, and other signs of neurological decline [6]. Cattaneo et al. [7] demonstrated a significant association between a higher dose of norepinephrine given and the occurrence of DSI. Cerebral vasospasm is a fundamental factor in the production of DCI and likely has a multifactorial origin. It is due to an imbalance in vasodilators, vasoconstrictors, and the stimulation of calcium [8]. In the walls of blood vessels, inflammation, and infiltration by leukocytes are prominent. Leukocytes promote the formation of free radicals, which can evoke endothelial dysfunction and calcium entry.

Furthermore, inflammation could be linked to elevated endothelin-1 levels, increased free radical formation, and reduced nitric oxide availability. The development of vasospasm is essential due to a relative or absolute excess of vasoconstrictor substances (such as endothelin-1) relative to vasodilator substances (such as nitric oxide). Other implicated theories include: (1) direct vasoconstrictor activity of extravasated blood degradation products, (2) development of structural changes within blood vessels, and (3) vasoconstriction through immune reactions. Several studies indicate that the development of DCI should not be entirely attributed to cerebral vasospasm and the role of microvascular spasm, cortical spreading ischemia, and microthrombosis due to coagulation cascade activation and fibrinolytic dysfunction also warrant consideration [8].

In addition to vasospasm, catecholamine excess following SAH has been implicated in BBB disruption, cerebral edema, and neuronal injury [9]. High catecholamine levels can increase BBB permeability by promoting endothelial damage, oxidative stress, and inflammatory cytokine release, leading to vasogenic cerebral edema [9]. This edema can exacerbate intracranial pressure and contribute to secondary brain injury edema [9]. Moreover, catecholamine-induced oxidative stress, mitochondrial dysfunction, and excitotoxicity can directly

cause neuronal injury and cell death, further impairing neurological outcomes in SAH patients [9]. These mechanisms highlight the multifaceted role of catecholamine surge in secondary brain injury following SAH beyond its contribution to vasospasm alone.

Cardiac Complications

Cardiac dysfunction after an SAH is common and is thought to result from the release of endogenous catecholamines [10]. The excessive release of catecholamines can lead to neurogenic stunned myocardium which is characterized by transient left ventricular dysfunction. It can also manifest as subendocardial ischemia, arrhythmias, and electrocardiogram changes in the absence of structural coronary artery disease [11]. Dinh et al. [12] reported that elevated catecholamine levels in SAH reduce cardiac output, impair myocardial function, and increase myogenic vasoconstriction in isolated cremaster arteries.

Furthermore, Naredi et al. [13] established that sympathetic nervous activation occurs in SAH and that the overactivation may relate to cardiac complications. The catecholamine surge theory has been linked to cardiac damage after both physical and emotional stressors [14]. The mechanism behind cardiac injury following SAH is thought to resemble that of apical ballooning syndrome (Takotsubo or stress cardiomyopathy) [10]. Stress cardiomyopathy is a complication of SAH, and its presentation initially is similar to acute coronary syndrome [15]. Studies have demonstrated a high incidence of stress cardiomyopathy in patients with SAH. As many as 28% of patients show signs of regional wall motion abnormalities, while up to 15% exhibit global left ventricular dysfunction accompanied by a reduced ejection fraction [15]. The exact pathomechanism of stress cardiomyopathy remains unclear, but it appears to be associated with catecholamine-induced myocardial stunning [15]. The main hypotheses for how catecholamine levels can lead to myocardial stunning are epicardial coronary vasospasm, acute microcirculatory dysfunction in the coronary arteries, and myocardial injury caused by catecholamines [15].

Release of excess endogenous catecholamines, along with various pathophysiological mechanisms, contributes to the development of stress cardiomyopathy in patients, with ranging severity [15]. The heightened sympathetic response and the increased myocardial sensitivity to catecholamines likely increase the risk of developing stress cardiomyopathy. The catecholamine-induced heart damage occurs through β-adrenoreceptors (βAR). At low-moderate levels, epinephrine boosts heart function via the β1AR-Gs pathway, and at high levels, it switches to the β2AR-Gi path, causing a reduced heart function; this is known as stimulus trafficking [15]. These levels activate a chain reaction that involves protein kinase A [15]. This enzyme affects calcium regulation, leading to calcium overload, mitochondrial dysfunction, oxidative stress, inflammation, and cell death. Stimulus trafficking can reduce these effects by switching to the β2AR-Gi pathway, which protects against cell death but weakens heart function, causing myocardial stunning and regional wall motion abnormalities [15].

In addition to stress cardiomyopathy, arrhythmias are common cardiac complications in SAH, occurring in 50%–100% of patients, and are associated with increased morbidity and mortality [16]. Atrial fibrillation, ventricular tachycardia, and QT interval prolongation can occur due to catecholamine surge, electrolyte disturbances, and autonomic dysfunction [16]. These arrhythmias increase the risk of hemodynamic instability, secondary cerebral hypoperfusion, and cardiac arrest [16]. Cardiac arrest, while less common, can occur in the acute phase of SAH, often due to ventricular arrhythmias or severe autonomic dysfunction, and is associated with poor neurological and overall outcomes [16]. Early recognition and management of arrhythmias and prevention of cardiac arrest are critical for improving prognosis in SAH patients.

Salem et al. [10] have shown that the left ventricular function changes parallel to catecholamine levels. The most abnormal echocardiographic findings are associated with the highest plasma levels of norepinephrine and epinephrine. Patients with left ventricular diastolic dysfunction exhibited significantly higher plasma norepinephrine levels compared to those without cardiac dysfunction [10]. Patients may also present with elevated troponin levels, B-type natriuretic peptide elevations, and other markers of cardiac stress, which correlate with SAH severity and complicate clinical management [10]. Robba et al. [17] state the importance of having timely medical care of both neurological and cardiac aspects of aSAH when it comes to improving outcomes and preventing further complications.

Metabolic and Systemic Effects

High levels of catecholamines can induce hyperglycemia, insulin resistance, and alterations in lipid metabolism, which

can impair recovery and increase the risk of secondary complications. Hyperglycemia is a marker of severity for SAH and is associated with systemic infections, vasospasms, and worst outcomes for patients [18]. Elevated catecholamine levels also contribute to fluid and electrolyte imbalances, including hyponatremia and hypokalemia, which are common in SAH patients [8]. Hyponatremia may result from cerebral salt wasting or the syndrome of inappropriate antidiuretic hormone secretion and can lead to worsened cerebral edema, increased risk of seizures, and poor neurological outcomes [19]. Hypokalemia can increase the risk of cardiac arrhythmias, particularly in the setting of catecholamine surge and QT prolongation, and may contribute to secondary brain injury due to impaired cellular function [20]. Excessive release of catecholamines is the principal cause of NPE, and it can result in irreversible injury to the hypothalamus and brainstem [8,21]. Inamasu et al. [22] showed that elevated norepinephrine may be more active in the SAH-induced NPE than epinephrine. Ogura et al. [23] suggested that sympathetic activation in aSAH cases is positively associated with the severity of the hemorrhage, immune and inflammatory response, and the development of vasospasm.

Catecholamine surge following aSAH has also been associated with the development of acute kidney injury (AKI) [24]. High catecholamine levels can lead to renal vasoconstriction, reduced renal blood flow, and ischemic tubular injury, contributing to AKI in these patients [24]. Additionally, catecholamine-induced hypertension and systemic inflammation may further exacerbate renal injury [24]. AKI in aSAH patients is clinically significant, as it is associated with increased morbidity, prolonged hospital stays, and worse neurological outcomes [24]. Early recognition and management of AKI in the context of catecholamine surge are essential to optimize patient outcomes and prevent secondary complications.

CLINICAL IMPLICATIONS

Monitoring and Diagnosis

Catecholamine levels and their metabolites in plasma or urine can be measured and used to assess the severity of the sympathetic response and the risk of complications. Dilraj et al. [25] reported that plasma catecholamine levels were significantly higher in patients with poor clinical outcomes than in those with better outcomes. Using these levels to determine which patients will have a poor clinical outcome may be possible. Continuous assessment of cardiac activity and blood pressure is critical in the acute phase of SAH. In terms of diagnosis, it is also essential to consider Pheochromocytoma. This is a catecholamine-secreting adrenal tumor that should be considered as a differential for hypertensive crisis and intracerebral hemorrhage [26].

Therapeutic Interventions

Beta-blockers and alpha-adrenergic antagonists have been explored for their potential to mitigate the adverse effects of endogenous catecholamine surge in aSAH. SAH increases myogenic vasoconstriction, and beta- and alpha-adrenergic receptors drive these cardiac and vascular effects [12]. Thus, these medications may help control blood pressure, reduce the risk of vasospasm, and protect cardiac function. In addition to blocking adrenergic activity, beta-blockers decrease cerebral metabolism and have cerebroprotective properties [15]. They can also prevent cardiac injury and normalize vascular function [12]. For example, cardiac dysfunction is entirely prevented by the use of bisoprolol, a B-adrenergic receptor antagonist. Furthermore, Dinh et al. [12] demonstrated that a1-adrenergic antagonism may be more effective than other medications commonly used and that prophylactic a1-adrenergic antagonism in high-risk SAH can help prevent delayed secondary increases in blood pressure. Hasegawa et al. [21] emphasize the need for further clinical studies to clarify the role of central sympathetic nerve activation in SAH.

The use of calcium channel blockers, such as Nimodipine, is well-established to prevent and treat vasospasm, although their interaction with catecholamine pathways is complex and warrants further study. Nimodipine is used to avoid the onset of DCI following an aSAH [6]. In addition to vasoconstriction and neuroprotection, Nimodipine is believed to decrease cortical spreading depression linked to DCI in aSAH [6,27]. It also seems to enhance fibrinolytic activity in aSAH patients by lowering plasminogen activator inhibitor-1 (PAI-1) levels, as high PAI-1 levels have been implicated in the pathogenesis of DCI [6]. It has been shown to improve overall outcomes of aSAH [8]. Other possible mechanisms of action include dilatation of tiny arteries, reduction of calcium-dependent excitotoxicity, and reduced platelet aggregation [8].

However, Nimodipine can cause a notable reduction in mean arterial pressure (MAP) and cerebral perfusion pressure (CPP) [6]. When this occurs, catecholamines are administered therapeutically as exogenous agents (e.g., norepinephrine) to maintain adequate MAP and CPP [4]. Hernández-Durán et al. [6] demonstrated that nimodipine interruption results in a higher incidence of DCI in patients with aSAH and that nimodipine interruption is a predictor of DCI in these patients. Rass et al. [27] have also shown that early recognition of drops in systolic blood pressure and the use of fluids and exogenous vasopressors, such as norepinephrine, is necessary to prevent hypotensive episodes. Because the incidence of DCI increases with nimodipine interruption, it is recommended to use vasopressors (exogenous catecholamines) first to treat hypotension and only, if that is ineffective, to reduce the nimodipine dose in half or stop entirely in the case of refractory hypotension [8]. Intravenous milrinone plus norepinephrine supplemented by intra-arterial Nimodipine has started being used as a rescue strategy for DCI following aneurysmal SAH. Intravenous milrinone combined with norepinephrine (exogenous catecholamine), along with intra-arterial nimodipine, has become a common approach for managing for DCI following aneurysmal SAH [28]. Steiger et al. [28] found that a treatment regimen involving milrinone and norepinephrine to induce hyperdynamic hypertension, combined with intra-arterial nimodipine spasmolysis when needed, results in significant improvement in cerebral perfusion during secondary cerebral ischemia after SAH.

Prognostic Value

Elevated catecholamine levels can be used as biomarkers for prognostication and stratification areas, as these are associated with worse outcomes, including higher mortality and long-term neurological deficits. Inamasu et al. [22] showed that patients with NPE sustained more severe SAH than those who did not experience NPE. Moreover, Hasegawa et al. [21] emphasized the significance of assessing central sympathetic nerve activation as a prognostic biomarker for SAH, noting its potential to guide decisions related to patient admission, treatment plans, and the use of supplementary medications to prevent further deterioration. Okazaki and Kuroda [29] also discussed using serum lactate measurements as markers reflecting the severity of sympathetic activity. Lång et al. [30] also stated that cardiac troponin measurements may improve assessments for long-term prognosis along with clinical risk scoring systems. An aSAH has a mortality rate of 25%–50%, but if prompt actions are taken, favorable outcomes are possible for aSAH [31].

CONCLUSIONS

Catecholamine surge can lead to hemodynamic instability, cardiac dysfunction, cerebral vasospasm, and altered metabolism, and can influence overall systemic inflammatory responses thus influencing the overall outcomes in patients with aneurysmal SAHs. There is a need for further research to develop targeted therapeutic strategies that can modulate the impact of catecholamine surge and thus change the outcomes favorably.

CONFLICT OF INTEREST

No potential conflict of interest relevant to this article was reported.

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Conceptualization: SN, TJ, LRMS, MY, AA. Formal analysis: SN, LRMS. Writing - original draft: SN, LRMS. Methodology:

SN, LRMS, AA. Project administration: SN, LRMS, AA. Writing - review & editing: SN, TJ, LRMS, MY, AA. All authors read and agreed to the published version of the manuscript.

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